



ANTI-DIABETIC ACTIVITY OF ETHANOLIC AND ETHYL ACETATE EXTRACTS OF *SOLANUM TRILOBATUM* LINN IN ALLOXAN INDUCED DIABETIC RATS

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ABSTRACT

The present study was under taken to evaluate the anti diabetic activity of *Solanum trilobatum* linn, solanaceae family. Ethanolic and Ethylacetate leaf extracts of *Solanum trilobatum* linn were prepared successively. The acute oral toxicity study were carried out for ethanolic and ethyl acetate leaf extract of *Solanum trilobatum* linn using fixed dose method according to OECD guideline number. Anti diabetic activity produced in ethanolic and ethyl acetate extract of *solanum trilobatum* increase the level of Blood Glucose, Cholesterol, Triglycerides, HDL

Key words: Anti diabetic, *Solanum trilobatum* Ethanol and Ethyl acetate.

INTRODUCTION

Diabetes mellitus is a metabolic disorder of multiple aetiology characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism due to deficient action of insulin on target tissues resulting, from defects in insulin secretion, insulin action or both (DeFronzo *et al.*, 1992; Chait and Brunzell, 1996)^{1, 2}. Diabetes mellitus may present with characteristics symptoms such as polyphagia, polydypsia, polyuria, blurring of vision and weight loss. Diabetes is a condition where the amount of glucose in your blood is too high because the body cannot use it properly. This is because your pancreas doesn't produce any insulin, or not enough insulin, to help glucose enter your body's cells – or the insulin that is produced does not work properly (known as insulin resistance). Insulin is the hormone produced by the pancreas that allows glucose to enter the body's cells, where it is used as fuel for energy so we can work, play and generally live our lives. It is vital for life. Glucose comes from digesting carbohydrate and is also produced by the liver. If you have diabetes, your body cannot make proper use of this glucose so it builds up in the blood and can't be used as fuel. There are two main types of diabetes: Type 1 diabetes and Type 2 diabetes.

Type 1 Diabetes Occurs abruptly, characterised by an absolute deficiency of insulin due to a marked decline in the number of insulin producing beta cells (perhaps caused by the auto immune destruction of beta cells) even though target cells contain insulin receptors. Type 1 diabetes is also known as insulin dependent diabetes and juvenile onset diabetes, as it most commonly develops in people under 20 years old though it persists through life, and requires periodic insulin injections to treat it. Although type 1 diabetes appears to have certain genes which make them more susceptible, some triggering factor is required e.g. viral infection, shock etc. Type 2 Diabetes It most often occurs in people who are over forty and overweight hence another name "maturity onset diabetes". Clinical symptoms are mild, and high glucose levels in the blood can usually be controlled by diet, exercise, and/or with anti diabetic drugs. Some type II diabetes have sufficient amounts of insulin in the blood, but they have defects in the molecular machinery that mediates the action of insulin on its target cells, cells can become less sensitive to insulin because they have fewer insulin receptors. 90% of all cases are type II.

MATERIALS AND METHODS: Collection of leaves: The plant leaves was collected during the month of January and was identified pharmacognostically by a botanist. Then powdered, weighed and stored in a clean, dry and air tight container. The powder was subjected to successive extraction with solvents n-hexane (n-hexane used to remove fatty substances) and ethanol.

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Extraction process: The defatted powder was packed in a round bottom flask and extracted with n-hexane at 60°C by Soxhlet apparatus to remove the oil content. After that the powder is extracted with ethanol at 60°C by condensation method. The above extract was dried on water bath at 70°C get a solid

$$\text{mass. \% Yield} = \frac{\text{Product obtained}}{\text{Weight of flowers taken}} \times 100$$

Animals: Adult male or female rat, weighing between 200 to 250 gm were used in the study. The study protocol was reviewed and approved by the institutional animal ethical committee and confirm to As per IAEC guidelines for the use and care of experimental animals in research. Animals were obtained from the shantiram college nandyal. Rats were housed in poly acrylic cages (38x23x10 cm) with not more 4 animals in cage. They were housed in an air conditioned room and were kept in standard laboratory condition under natural light and dark cycle are maintained humidity 60±5% and an ambient temperature of 25±2°C. All experiments were performed between 9.00 am to 4.00 pm. The animals were free access to standard diet and tap water is allowed to acclimatize for one week before the experiments. Commercial pallets diet contained 22% proteins, 4% fat , 4% fibre , 36% carbohydrates and 10% ash w/w are supplied.

STATISTICAL ANALYSIS

Anti diabetic data were expressed as mean ±S.D and evaluated by ANOVA

EXPERIMENTAL DESIGN

Animals were divided in to five groups of six rats each. Group-I :normal rats administered distilled water ,2ml/kg,orally daily for 10 days .Group-II : diabetic control rats administered 0.2ml of 2% aqueous gum acasia.Group-III :diabetic rats administered EEST,100mg/kg, orally daily for 10 days.Group-VI: diabetic rats administered EEST,200mg/kg,orally daily for 10 days.Group-V:diabetics rats administered standarad drug glibenclamide (10mg/kg,orally) daily for 10days.Body weights of experimental rats were measured on days 3,5,7and 10.after 10 days of treatment, all the rats were anaesthetized and sacrificed by cervical dislocation

EXPERIMENTAL

In the present study albino Wister rats weighing about 200-250 gms were selected and divided into 6groups containing 6 animals in each group.

S. no	Group	Treatment	
1	Group A	Standard	Administered distilled water 2.0ml/kg orally
2	Group B	0.2ml of 2%	Administered 0.2ml of 2% aqueous gum acacia orally
3	Group C	100mg/kg	Administered 100mg/kg EAEST orally
		200mg/kg	Administered 200mg/kg EAEST orally
4	Group D	100mg/kg	Administered 100mg/kg EEST orally
		200mg/kg	Administered 200mg/kg EEST orally
5	Group E	10mg/kg	Administered standard drug glibenclamide 10mg/kg,orally

Calculation

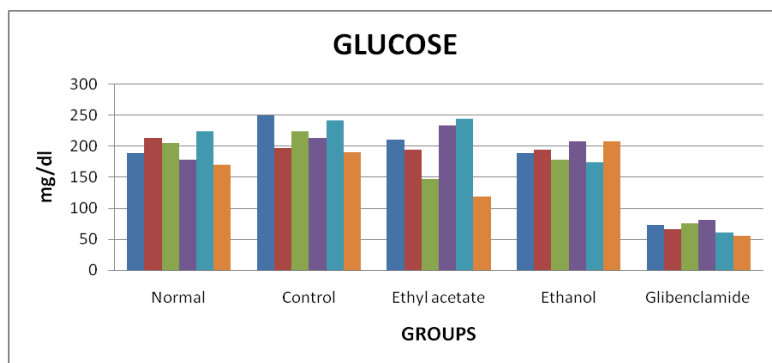
$$\text{Concentration of Sample (mg/dl)} = \frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{concentration of standard 100mg/dl}$$

RESULTS

The effect of EEST and EAEST of Blood glucose

S. no	Normal	Control	Ethyl acetate	Ethanol	Glibenclamide
1	189.47	250	210.52	189.47	73.63
2	213.15	197.36	194.76	194.73	65.78
3	205.26	223.68	147.36	178.94	76.31
4	178.94	213.15	234.21	207.89	81.57
5	223.68	242.1	244.73	173.68	60.52
6	170.54	190.0	118.42	207.89	55.5
Mean± S.D	202.1±1.2	225.25±1.4	191.66±1.6	192.1±1.8	71.56±2.0

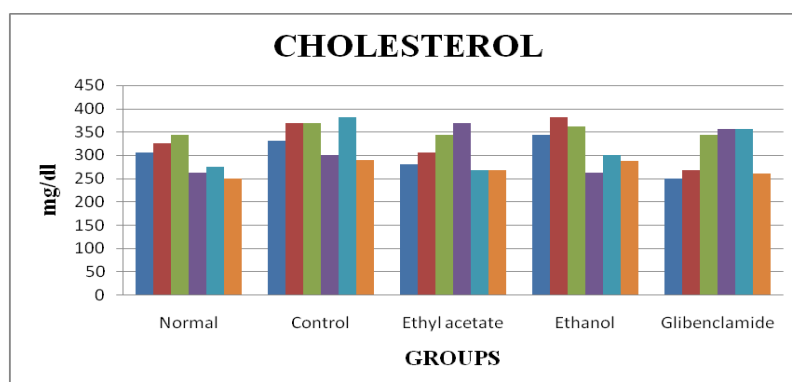
Results are expressed as Mean ± S.D. All the groups are compared with control. n=6 and p value is <0.0001



The effect of EEST and EAEST of cholesterol

S.NO	Normal	Control	Ethyl acetate	Ethanol	Glibenclamide
1	306.25	331.25	281.25	343.75	250
2	325	368.75	306.25	381.25	268.75
3	343.75	368.75	343.75	362.5	343.75
4	262.5	300	368.75	262.5	356.25
5	275	381.25	268.75	300	356.25
6	250.2	290.35	268.75	287.5	260.15
Mean±sd	302.5±2.0	350±2.4	306.25±1.8	322.91±1.2	315±1.2

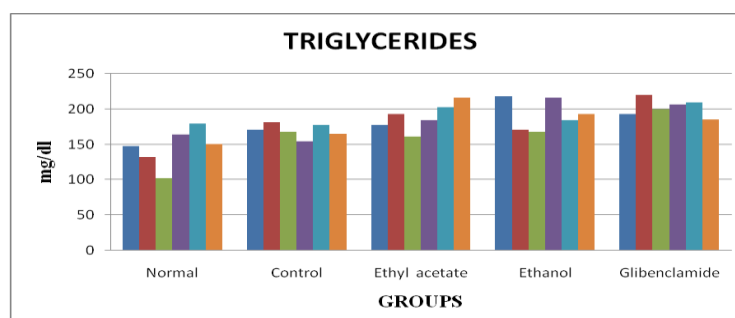
Results are expressed as Mean ± S.D. All the groups are compared with control. n=6 and p value is <0.0001



The effect of EEST and EAEST of Triglycerides

S.NO	Normal	Control	Ethyl acetate	Ethanol	Glibenclamide
1	147.7	170.45	177.27	218.18	193.18
2	131.81	181.81	193.18	170.45	220.45
3	102.27	168.18	161.36	168.18	200
4	163.63	154.54	184.09	215.9	206.81
5	179.54	177.27	202.27	184.09	209.09
6	150.50	165.25	215.90	193.18	185.15
Mean±S.D	144.99±1.2	170.45±1.6	189.01±1.8	191.66±1.4	205.9±1.2

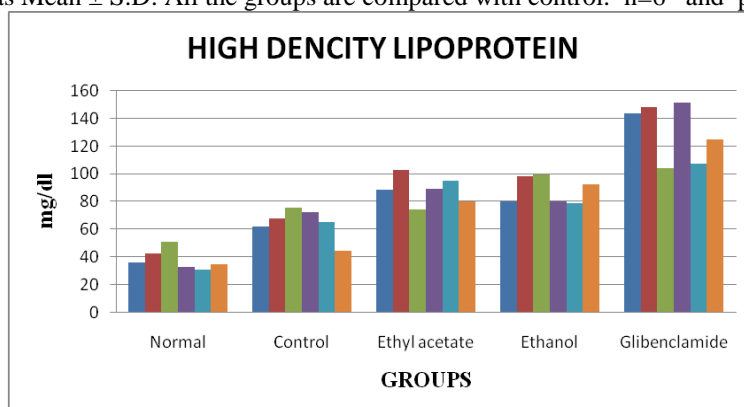
Results are expressed as Mean ± S.D, All the groups are compared with control. n=6 and p value is <0.0001



The effect of EEST and EAEST of HDL

S.NO	Normal	Control	Ethyl acetate	Ethanol	Glibenclamide
1	36	62.12	89.039	80.3	143.93
2	43	68.18	103.03	98.48	148.48
3	51	75.75	74.24	100	104
4	33	72.72	89.39	80.3	151.51
5	31	65.15	95.45	79.24	107.57
6	35	44.5	80.3	92.42	125.12
Mean±sd	58.8±1.6	68.78±1.2	88.63±1.4	87.62±1.6	131.098

Results are expressed as Mean ± S.D. All the groups are compared with control. n=6 and p value is <0.0001



DISCUSSION

Pancreas is the primary organ involved in sensing the organisms dietary and energetic status via glucose concentration in the blood and in response to elevated blood glucose, insulin is secreted. Alloxan is one of the usual substances used for the induction of diabetes mellitus apart from streptozotocin. Alloxan has a destructive effect on the beta cells of the pancreas. Alloxan a beta cytotoxin, destroys beta cells of islets of langerhans of pancreas resulting in a decrease endogenous insulin secretion and paves the ways for the decreased utilization of glucose by the tissue. It results in elevation of blood glucose level. Expression of elevated fasting blood glucose level conformed induction of diabetes in alloxan induced experimental rats, there by inducing hyperglycemia. Insulin deficiency leads to various metabolic alterations in the animals viz increased blood glucose, increased cholesterol, increased levels of alkaline phosphate and transaminases. Experimental studies reveal that ethanolic and ethyl acetate extract from *Solanum trilobatum* (100,200 mg/kg) orally administered for 10 days produce a significant increase in blood glucose level in the model of alloxan induced diabetes in rats. It also proves the traditional claim with regard to *Solanum trilobatum* for its anti diabetic activity.

CONCLUSION

In the present investigation it was observed that diabetes produced by the alloxan is prevented by treatment of groups of Ethanolic and Ethyl acetate extracts of *Solanum trilobatum* decrease the levels of blood glucose, cholesterol, triglycerides, high density lipids (HDL). This protective effect of

ethanolic and ethyl acetate extracts may be due to the presence of saponins & tannins. Further studies are needed to elucidate the particular saponins and tannins which are responsible for anti diabetic activity.

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